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TWO CASES OF RELAPSING FEVER; WITH NOTES ON THE OCCURRENCE OF THIS DISEASE THROUGHOUT THE WORLD AT THE PRESENT DAY.*

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INTRODUCTION.

THE first of these two cases, exceptional not only in its occurrence but in some of its clinical features also, shows that other cases occur from time to time which are mistaken because of the mildness of the symptoms or insufficient care in examining the blood.

There are no other cases on record in this country up to the present time in which *Sp. Obermeieri* has been found in the blood.

The origin of the first case has not been directly traced, but the existence of cases in tropical America, hitherto unsuspected, seems to indicate its source and makes its appearance here less surprising.

The second case was the result of accidental inoculation.

CLINICAL HISTORY.

CASE I.—C. N., 39 years of age, an Englishman and by occupation a ship's steward, walked into Bellevue Hospital at 9 A. M. on August 15, 1905, and was admitted to the Third Medical Division.

Previous history.—He arrived in New York on August 11, 1905, on a steamer of the Galveston Line plying between New York, Galveston, Tex., and Key West, Fla. He had served three trips on this steamer as steward, being five days at sea and three days in Galveston each trip. He knew of no cases of illness on board nor in Galveston nor in his boarding house in New York. Prior to this he had spent six

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months in Santo Domingo and Porto Rico, had made several voyages to South America, and the West Indies during the last five years, and had not been sick during this time. Most of his sea life previously had been spent between England, China, and the East Indies. Twelve years ago (1893) while in the coasting trade out of Bombay and Calcutta had malaria (?) fever. The attack lasted six weeks. He never had had typhoid fever nor syphilis.

Present illness.—On August 13, 36 hours previous to admission, he had a distinct chill lasting half an hour, and following the chill some sweating, continuing probably an hour and a half. He had severe frontal headache and did not sleep well that night. On the next day, though he had no chill, he suffered from chilliness, headache, and thirst. That night he felt weak and could not sleep and concluded to go to the hospital the next day. From the 12th he had had three thin stools each day; no vomiting; no epistaxis; no cough.

On admission his rectal temperature was 102.2° F. and pulse 96, regular and of good force, the chief complaints being severe frontal headache, general aching pains, and some stiffness of the legs. On his face, neck, and upper part of trunk there was an erythematous blush; a few very small pink macules which disappeared on pressure and returned were seen on the trunk. Mucous membranes appeared normal; lymph nodes in neck and groins were just palpable. Tongue was moist, broad, slightly tremulous, with slight brown coating. The spleen was palpable and somewhat tender on pressure. There was little tympanites and some slight tenderness over the descending colon. A few sibilant râles were heard over both lungs. There were no other objective symptoms nor abnormal signs. He was a spare man, fairly well developed and nourished, and weighed 130 to 135 lbs. He was placed on a milk diet at first. Medication throughout his run of fever was placebo.

During the afternoon of the day of admission patient had a slight chill followed by a fall of temperature to 98.5° at 1 A. M. of the 16th. The leucocyte count was 7,800, Widal reaction negative, 1 to 10. (The differential blood counts are given in the table below.)

August 16. Temperature at 3 P. M., 101.6°, pulse 92, respirations 24. No malaria parasites seen on careful search. Urine: 30 oz., sp. gr. 1014, faint trace of albumin, few small hyaline casts; no diazo-reaction. The temperature declined steadily after 3 P. M. to 96.4° at 5 A. M. on 17th, the pulse receding to 52. Patient fairly comfortable with hot bottles and blankets. Has had two or three loose stools during the past two days.

On August 17, 18, and 19 patient was comfortable, no headache, no abdominal tenderness or distension; stools formed. Spots have disappeared. Spleen is palpable. The pulse reached 48, temperature subnormal.

August 20 (seventh day). At 3 P. M. temperature 98.5° F., pulse 56. The second paroxysm now began, the fever rising steadily until by noon of 21st it reached its highest point, 104.4° F., pulse 96, respirations 24. Bowels constipated, headache, shiverings, and general discomfort. Total urine 44 oz.

Examinations for malaria parasites were unsuccessful. Temperature declined sharply (97° F. by 9 P. M.), with some sweating. Pulse 52, respirations 18.

August 24, 9 P. M. (three days after last paroxysm). The temperature which had been subnormal for three days rose to 100.2° F., pulse 88, declined quickly to 97.2° F., pulse 68, and by midnight of 25th had reached 96.5° F., pulse 56. Meanwhile the spleen remained enlarged, bowels constipated. There was no eruption. Three

days later, 27th, a similar rise to 99.6° with acceleration of pulse was noted, and disturbed sleep.

With these two exceptions the temperature range was subnormal almost continuously (a few times reaching 98.5° F.) for nine days until 6 P. M. of August 30. Patient was out of bed and on regular diet. During this interval the patient's temperature was taken every three hours and daily searches were earnestly made for the malaria hemamœba by every member of the staff, all without success. It was this persistent scrutiny, however, that was finally rewarded by the discovery by the house physician, Dr. Heitlinger, of spirochetes in a stained smear made just after noon of August 31—temperature 102.4°. He found six in one smear ($\frac{1}{12}$ immers., oc. II).

This, the third distinct paroxysm, began at about 6 A. M. August 30, and continued for 36 hours. At the beginning he asked for an extra blanket, but he had no distinct chill at any time during the pyrexia. He had severe headache and said he felt "mean all over;" he says he can tell when the attack is coming on because he dreams the night before and is wakeful.

The spleen is tender and more easily palpated. During the decline of temperature there was a return of the looseness of bowels; five stools. Blood taken during night of 31st and forenoon of September 1 contained about six spirochetes to each smear. On September 2, 24 hours after the crisis, one spirochete was found after careful scrutiny of four smears. Highest temperature of this paroxysm was 103° F. Crisis occurred in early hours of September 1, and by 9 A. M. the temperature was 98.5°.

The pulse ran higher during this paroxysm than in first two (112 to 118). Promptly after the crisis the patient felt well, diarrhea ceased, splenic tenderness subsided but the spleen remained palpable.

On and after September 2, the patient was up and about every day during the rest of his stay in hospital (till November 9) except at the time of the fourth and last period of pyrexia (September 9 to September 11). A constant watch was kept every three hours on his temperature until October 6. Weight September 2, 130 lbs.

There is nothing notable of this last intermission except that the spleen remained palpable and patient's weight increased to 134 lbs. His appetite improved; he felt well. He had been placed on a tonic of Fe et Potas. Tart. and Port wine.

September 9. During night of September 9 and 10 patient was restless and could not sleep; had severe headache; felt cold but had no chill; temperature at 5 A. M., 97.6° F., pulse 80, respirations 28. Fever began and by midnight of 10th reached 103° F., pulse from 112 to 120. Face flushed, skin hot and dry, erythematous blush appeared as in the first paroxysm. Spleen enlarged and tender; bowels loose. A remission of 2.5° F. occurred during night with some lessening of pulse rate, but at 9 A. M. with a temperature of 100.4° F., pulse was still 108.

The temperature then rose steadily during the afternoon, reaching the highest point of the fever at 7 P. M., 104.5° F., the patient meanwhile being greatly distressed with frontal headache and muscular pains, and had a pretty severe chill. Pulse 124. Crisis was suddenly ushered in at 7 P. M. by sweating and fall in temperature (7.5° in 10 hours) and coincidentally marked amelioration of all the symptoms. Increased looseness of bowels was noted. Weight 131 lbs., a loss of three pounds during the paroxysm. September 13, complained of considerable pain in right shoulder. No objective signs.

SPIROCHETES.

Sept. 10	2 P. M.	Temp.	101°	1	in	13	smears
	10	"	102.8	3	"	2	"
	12	"	103	7	"	2	"
Sept. 12	11 A. M.	"	98	None	"	4	"
Sept. 15	11	"	98.8	"	"	4	"

From this time on convalescence was uneventful. The temperature ranged between 97° F. in the early morning and 98.5° in the evening until the 25th, after that it ran about normal until patient's discharge on November 9, except that several times it rose to 99.6 °F. as on the 19th and 20th (nine days after last paroxysm) and on the 28th, when patient reported as being restless and uneasy. Nothing untoward developed though the spleen could be felt. By October 1, he had pretty well recovered from his anemia, the differential count was nearly normal. No spirochetes could be found. The spleen could not be felt. On discharge patient weighed 152 lbs.*

Each attack of fever that was observed was characterized by prodromic wakefulness and restlessness; chilliness, rapid, though not abrupt, rise of temperature beginning in the forenoon; decided increase of pulse-rate compared with the interval; severe headache moderate enlargement of spleen; high percentage of large lymphocytes (and in the first and fourth paroxysms by an erythematous rash) and ended in a crisis, the rapid fall in temperature to subnormal occurring during night hours with some diarrhea, sweating, and marked fall in pulse-rate. No jaundice was observed nor gastric symptom at any time. The features of the intervals were subnormal temperature, slow pulse, and rapid return to a feeling of well-being. The patient was not very sick at any time.

Spirochetes.—The organisms found were to all appearances identical with *Sp. Obermeieri.* The accompanying photographs (1500 diam.) furnish the best description, all three of which were taken from the same microscopic slide. The organisms were not very numerous. They were first seen in the stained specimen (Wright's stain). They varied in form and size; they appeared in increasing numbers during the access of fever and disappeared rapidly after the crisis. Only once were any seen during the interval, as is noted above. This was about 36 hours after the crisis. Three days later the unsuccessful inoculation was made.

No cases developed in the ward or hospital as a result of the

*I take this opportunity of thanking Drs. Heitlinger, Rimer, and Satchwell, of the housestaff for their unremitting care and attention given in this case.

presence of this case which was treated in general ward, no special means being taken to prevent infection.

CASE I.

URINE EXAMINATIONS.

	AUGUST					
	16	17	19	21	29	31
Stage of Disease	1st Parox'm	Day after Crisis	1 Day before Parox'm	2d Parax'm	1 Day before Parox'm	3d Parox'm
C.c. in 24 hours	887	1,124	1,124	1,301	887	1,124
Sp. Gr.	1,014				1,010	
Urea 24 hrs., grammes					7.77	
Reaction	Ac.				Ac.	
Albumin	Faint Tr.				None	
Sugar	None				None	
Diazo-	None					
Microscope	A few small hyaline casts				A few small hyaline casts	
Diet	Milk	Milk	Milk	Milk	Unrestricted Diet	

	SEPTEMBER					OCTOBER
	1	7	9	11	12	2
Stage of Disease	Day after Crisis	3 Days before Parox'm	1 Day before Parox'm	4th Parox'm	Day after Crisis	21 Days after Crisis
C.c. in 24 hrs.	1,360	2,070	2,839	1,715	2,129	1,479
Sp. Gr.		1,015	1,014		1,012	1,010
Urea 24 hrs., grammes		22.68	27.99		20.99	9.72
Reaction		Ac.	Ac.		Alk.	Neut.
Albumin		None	None		None	None
Sugar		None	None		None	None
Diazo-						
Microscope		Neg.	Neg.		Cryst. and Amorph. Phosph.	Neg.
Diet	Unrestricted Diet					

On September 5, four days after a paroxysm and three days after the last spirochete was found, a portion of 7 c.c. of blood drawn from the arm of this patient was injected directly subcutaneously into a monkey (*Macacus rhesus*). There was no reaction. On September 10, during the access of the last paroxysm, 5 c.c. was withdrawn from the patient and 3 c.c. injected subcutaneously.* It

* The further results of this and other animal inoculations are reported by Dr. Norris and his assistants, Drs. Pappenheimer and Flournoy, in the article that follows in this number of the *Journal*.

was during the manipulations incident to these experiments that the second case developed. The clinical account of this I present in the words of the patient himself.

CASE II.—The patient was one of the writers of the article that follows. Infection probably took place through being bitten by one of the monkeys, during the latter's paroxysm. After the fracas incidental to having their temperature taken, the monkeys frequently bled from the gums, and it is reasonable to suppose that spirochete blood was inoculated with the bite in this way. This may have occurred on one of several occasions, and therefore the exact period of incubation cannot be made out with certainty.

Family and previous history have no bearing upon the case, and may be omitted.

On the afternoon of October 8, the patient was suddenly taken ill with chilly sensations, severe frontal headache, shooting pains in the extremities, and moderate general prostration. This increased during the night, and the following afternoon he took to this bed. Appetite was completely lost from the beginning. There was no nausea or vomiting, and no icterus; bowels constipated.

The temperature began to rise about 12 hours after the onset of the subjective symptoms, reaching 103° on the afternoon of the second day. It then fell to 100° and remained comparatively low until the fourth day, when it reached 105°; this was followed after a few hours by a critical defervescence; marked by a drop to normal within 12 hours. The first paroxysm, therefore, lasted four days. Following the crisis there was an apyrexial period lasting for seven days, during which the temperature was persistently subnormal, ranging from 95° to 98°. During the interval there was an absence of all subjective symptoms. The patient was out of bed on the second day, regained strength and appetite rapidly, and was completely recovered before the onset of his first relapse.

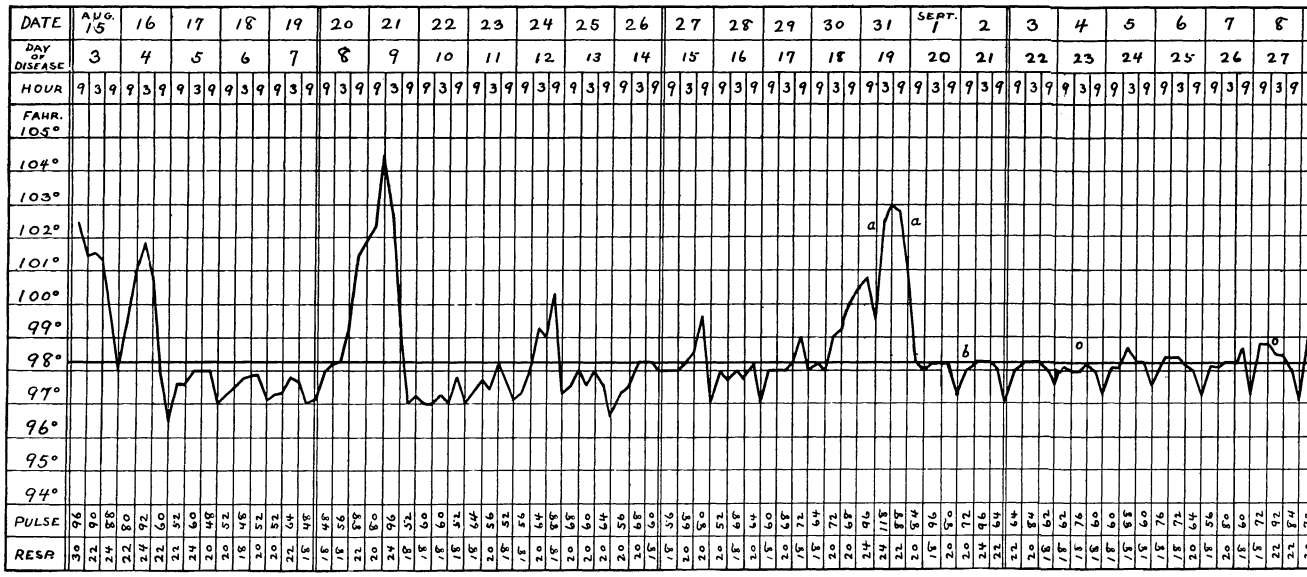
On October 18, seven days after the first crisis, there was a recurrence of the subjective symptoms marking the onset of the first attack. After a prodromal period of about 12 hours, the temperature again rose, reaching 102° on the afternoon of October 19. During the ensuing four days, there was remittent pyrexia. On the morning of October 21, the temperature fell to normal, to rise again to 101.8° during the day. The following morning it fell to 95°, then rapidly rose, and at 8 P. M. reached its highest point, 105.4°, to fall by crisis to subnormal during the night. These remissions in the temperature were attended by a transient release from all feelings of illness and discomfort.

The second paroxysm, therefore, lasted for four days. There succeeded a second apyretic interval of eight days.

At the end of this period there was a recurrence of the usual symptoms, accompanied by a febrile paroxysm lasting two days. On the morning of the second day, there was a remission to normal, followed by an abrupt rise to 105°, and an equally abrupt crisis during the following night.

The third paroxysm, therefore, lasted for but two days. The temperature remained subnormal for several days, then gradually rose to normal. Convalescence after the third attack was rapid, and progressive. Only for a few hours, on the 12th day after the final crisis as there a recurrence of the sense of nervousness and malaise

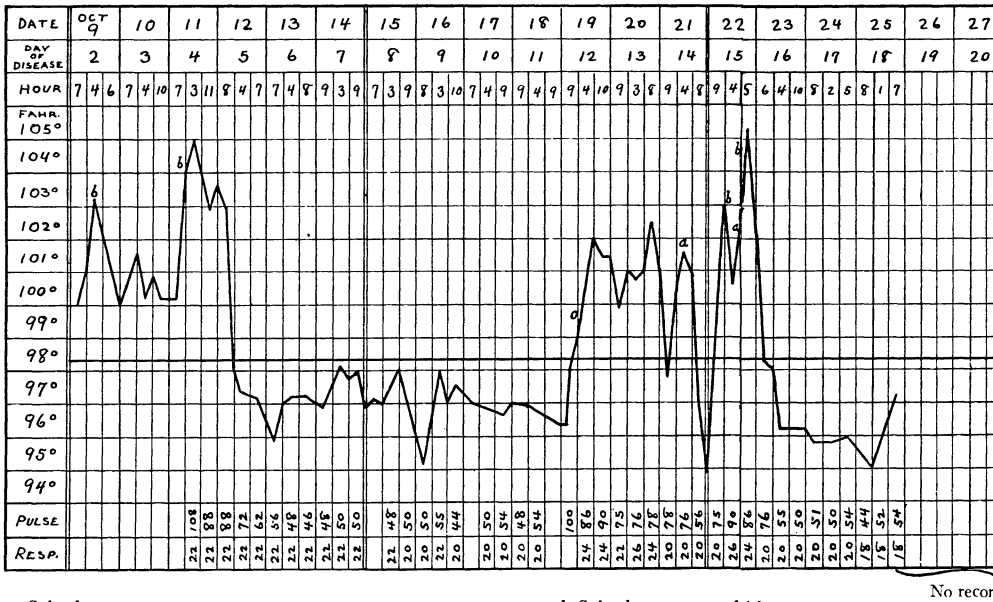
RELAPSING FEVER, CASE I, C. N., BELLEVUE HOSPITAL, August 15, 1905.



No malaria organisms in daily search.

a, 6 spirochetes in 1 smear.
b, 1 " " 4 smears.
c, 1 spirochetes in 13 smears.
d, 3 " " 2 "

RELAPSING FEVER, CASE II, October 8, 1905.

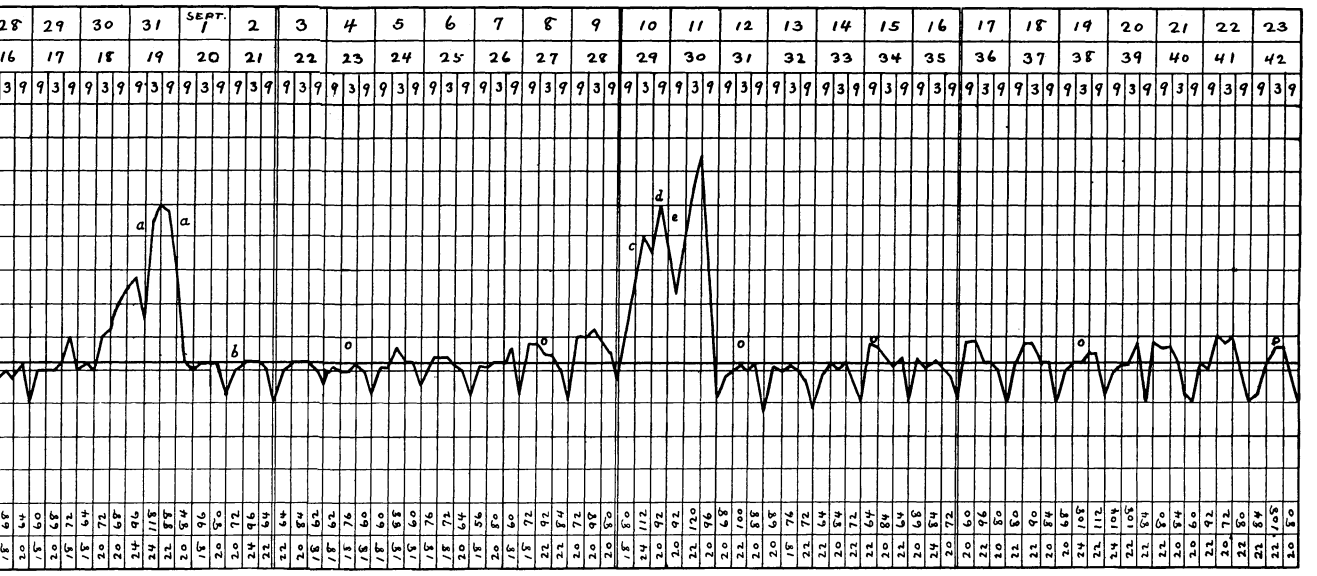


a, Spirochetes present, not numerous.

b, Spirochetes present, fairly numerous.

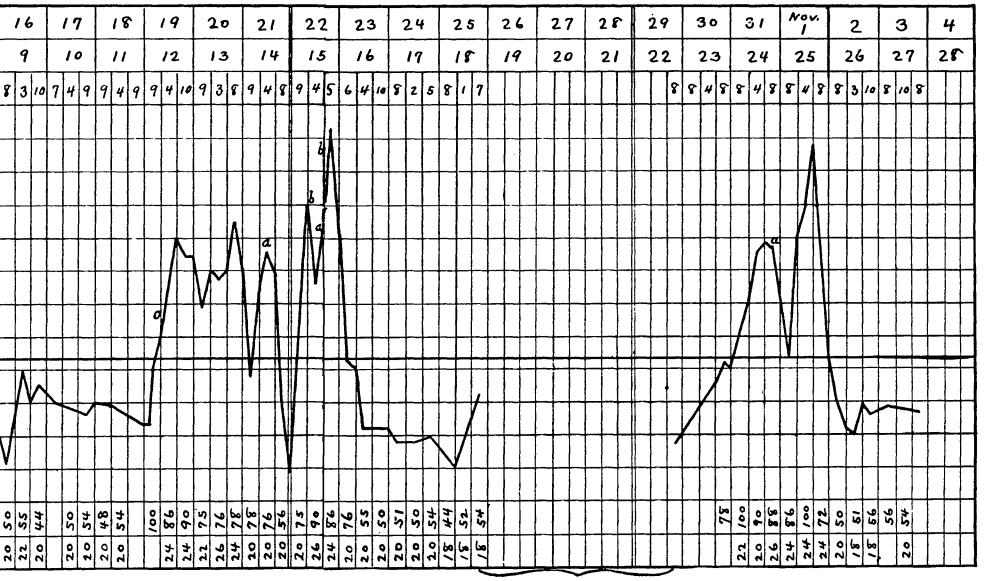
No recor

15, 1905.



c, 1 spirochetes in 13 smears.
d, 3 " " 2 "
e, 7 spirochetes in 2 smears.
o, No " " 4 "

8, 1905.



No records taken.

b, Spirochetes present, fairly numerous.
o, Spirochetes absent.

marking the onset of the previous attacks. There was no febrile reaction, however, and this may have been an incidental condition unrelated to the illness.

The course of the disease as outlined above is fairly typical of the relapsing fever caused by the *Sp. Obermeieri*. The duration of the febrile paroxysms is somewhat shorter than the average; yet great variations in the length of the pyrexial and apyrexial periods are known to occur.

Certain individual symptoms may be commented upon. The pulse-rate throughout the febrile period remained relatively slow, as compared with the temperature. It rarely exceeded 100; ranged usually about 80, and on one occasion with a temperature of 105.4°, was only 76. A marked bradycardia accompanied the subnormal temperature between the paroxysms, the pulse-rate ranging from 40 to 50.

Chilly sensations were frequent throughout the greater portion of the fever; during the first relapse, with a sudden rise of temperature, there was a severe, shaking chill which lasted about six hours. This was the only real chill that occurred.

Darting pains in the limbs, and especially in the inguinal regions, were present with each attack, and at times were quite distressing. A dull pain in the back, and in the splenic region was also felt at times.

The sudden falls of temperature were attended by very profuse sweating, usually coming on during the night. These drenching sweats also persisted for several nights after each crisis.

The spleen, examined daily, was palpable only once (October 20, first relapse), and did not appear to be much enlarged. Slight edema about the eyelids was seen in the early mornings, but disappeared after a few hours.

The urine showed a trace of albumin and a few granular and hyaline casts. The excretion of urea was increased. The diazo-reaction was negative.

The blood naturally showed the most interesting changes. Spirochetes were present during each febrile period, disappearing after the crisis. They were never numerous, however, and during the last paroxysm were found but once out of four examinations, and in very small numbers; this, too, in spite of the high temperature and severe constitutional symptoms.

The leucocytes counted on October 9, the second day of the illness, were 14,400. Unfortunately no subsequent counts were made.

A marked and striking relative, and probably also an absolute, increase in the number of large mononuclear cells, or megacaryocytes, occurred with each attack. This is well shown by the following differential counts, made at various times during the illness. Transitional forms and degenerated polymorphonuclears were also in excess during the febrile period.

Aside from this the blood showed little of interest. Polychromatophilia of any degree or granular degeneration was not present.

Convalescence after the second relapse was exceedingly rapid, and save for slight loss of weight and weakness after severe exertion, the patient was in normal health within a week after his final crisis.

Treatment was entirely symptomatic, and apparently in no way influenced the course of the illness.

RELAPSING FEVER.

BLOOD RECORDS.*

CASE I. 300 WHITE CELLS COUNTED.

Day of Disease.	3	19	21	23	27	30	38	50
Stage of Disease..	1st Par'x'm 3d Day	3d Par'x'm 1st Day	3d Inter. 2d Day	3d Inter. 4th Day	3d Inter. 8th Day	4th Parox'm 2d Day	Last Apyrex'l Period	Last Apyrex'l Period
Time and Temperature.....	1–3 P. M. 101.5°	1–3 P. M. 102.5°	1–3 P. M. 98.2°	1–3 P. M. 98°	1–3 P. M. 98.8°	1–3 P. M. 103°	1–3 P. M. 98.4°	1–3 P. M. 98.6°
No. of reds.....				4,100,000[1]		3,400,000	3,600,000	4,480,000
No. of whites ...	7,800			5,600		5,700	7,600	7,600
Ratio.....				1:732		1:596	1:473	1:589
Hb % (Tal'quist)				80		70	70	90+
Polymorph.....	73%	50%	54.2%	55.6%	59.6%	62.1%		64%
Small lymph	25	17.3	10.8	7.2	2.3	5.5		33
Large lymph		27.5	30.6	31	27.9	24.5		
Mononuc. and transition	1	5	4.2	5	5.7	5.5		1.5
Eosinophile.....	0.5	0.1		1	3.5	1		0.5
Mast cells	0.5	0.1	0.1	0.1	1	1		1

CASE II. 100 WHITE CELLS COUNTED.

Day of Disease..	2	6	12	14	15	24	25	25
Stage of Disease..	1st Parox'm 2d Day	1st Inter. 1st Day	2d Parox'm 1st Day	2d Parox'm 3d Day	2d Parox'm 4th Day	3d Parox'm 1st Day	3d Parox'm 2d Day	3d Parox'm 2d Day
Time and Temperature.....	P. M. 103°	P. M. 97°	12 M. 99°	P. M. 101°	P. M. 103°	11 P. M. 100.8°	1 P. M. 101.8°	7 P. M. 104.8°
No. of reds								
No. of whites ...	14,400							
Ratio.....								
Hb % (Tal'quist)								
Polymorph.....		40%	71%	61%	68%	67%	71%	79%
Small lymph	Incre'd	6	6		3	3	4	1
Large lymph		9	16	3	1	9	1	2
Mononuc. and transition		43	4	35	32	21	24	17
Eosinophile.....		2	4	1	1		0.	1
Mast cells								

SUMMARY.

	Case I	Case II
Duration of paroxysms	3, 1½, 1½, 2 days	4, 4, 2 days
Duration intermissions	3, 9, 9 days	7, 8 days
Highest rectal temperature recorded	105.5° in last relapse	105.4° in first relapse
Lowest rectal temperature recorded	96.4° in first crisis	94.8° in second crisis
Highest pulse-rate recorded	124 in last relapse	110 in second relapse
Lowest pulse-rate recorded	48 in first crisis	44 in first and second crises
Greatest fall in temperature at crisis	7.5° in 10 hours	8.8° in 17 hours
Greatest fall in pulse-rate at crisis	124−68=56 at last crisis	110−47=63 in last crisis

* All the blood-counts of Case I were made by Dr. Satchwell of the interne staff; those of Case II by the patient himself.

[1] Marked granular degeneration of the red cells.

On a cursory view of the temperature and other features of Case I, there is nothing suggestive of relapsing fever to anyone not well acquainted with this disease, and even then he might miss the diagnosis, for so good an observer as H. Vandyke Carter, speaking of the first cases he saw in 1885–86, says, "without special attention it is always possible that genuine spirillum fever may be confounded under 'Remittents.'" But when a spirochete is found in the blood which bears a distinct relation to the paroxysms and is identical in its morphology to that described by Obermeier, and when a second case develops by infection from this one which strongly resembles the typical descriptions of relapsing fever, one can hardly avoid the conclusion that the disease in question is really one of true relapsing fever.

The paroxysms in both cases resemble those of relapsing fever in the abrupt onset with chill or chilliness; the frequent recurrence of this symptom just before the crisis at the acme of the attack; the moist skin and sweating during the febrile periods; and in Case II drenching sweats during, and for some time subsequent to, the crisis; the moist tongue—this is mentioned by Murchison as being the rule throughout the attack, and Flint says it is somewhat distinctive of this disease. The severe headache, muscular and leg pains are very characteristic, and arthritic pains without inflammatory or other joint symptoms not infrequently occur. The moderate enlargement of the spleen and the splenic tenderness are other points of resemblance. And lastly, the crisis occurring during the night time far more frequently than during the day time (Lebert, Carter, Strümpell, and others). The intermissions were marked by extraordinarily low temperature and pulse records, especially in Case II, and this for days at a time.

Variations occur in this disease, as Strümpell says, as well as in any other disease, and it is this fact that Case I emphasizes.

SUMMARY OF CERTAIN CLINICAL FEATURES OF RELAPSING FEVER.

The duration of the initial paroxysm.—Murchison says in rare instances it does not exceed three or four days; Flint says, exceptionally, it may be only two days; Carter says it may end at any time after the fourth or fifth day.

The duration of the intermission.—The longest Murchison says was 12 days; it may not exceed two or three days, and it may extend to 12 days or more (Flint); sometimes only four or five days, rarely two weeks or so (Lebert, who believed relapsing fever and bilious typhoid were not identical); from 6 to 12 days and may extend to 15 days (Sandwith).

Duration of relapses.—Many instances could be given of variation in length from a few hours up to, and even longer than, the primary fever.

Number of relapses.—Murchison says five may occur "making in all six paroxysms;" Flint never saw more than one; Lebert says: "I had not formerly believed in a fourth relapse until during the last epidemic several cases presented it undeniably;" and, finally, Strümpell says the more accurately and persistently we take the temperature during convalescence, the oftener do we find slight rises of temperature occurring at intervals late in the history of the case (see chart of Case I and note the remark in history of convalescence of Case II).

Remissions in the temperature.—Often there are oscillations to a considerable degree (Strümpell). In Carter's 270 cases in 1876–78 pseudo-crises were remarkable in both invasion and relapse, and in his cases in 1885–86 he says the perturbations of temperatures were marked. Other instances might be cited occurring both before and after Obermeier's discovery.

It should be noted that we can hardly compare the charts of these two cases to those in the textbooks in this respect, as the former represent three or four hour intervals, while the latter generally show only morning and evening variations.

Pulse-rate.—Usually high in the paroxysms, it may vary from 90 to 110, according to Murchison, but he also says there is less correspondence between the pulse and the temperature in the relapse than in the first paroxysm, instancing a temperature of 106° F. with a pulse of 90 (see Case II of this report—temperature 105.4°, pulse 76).

Nausea and vomiting are among the most common symptoms, sufficiently so, as Flint says, to be somewhat distinctive, especially when the disease is contrasted with typhus and typhoid fevers.

In our two cases they did not occur in the first case, but in the second there was some nausea shortly after the crisis. Nor did the great enlargement of the spleen, reported by some, exist in our cases. The spleen as a rule, however, seems to be but moderately enlarged.

Our two cases differed as regards the condition of the bowels; Case I had diarrhea frequently, and in this case sweating was much less prominent than in Case II.

These two cases then show considerable similarity to the clinical history of relapsing fever as presented by the older writers and the variations which do occur are within the limits of variation recognized by them. Care should be taken that a sporadic instance of disease be not made to conform with earlier descriptions, and, on the other hand, the case in question should not be classified as a new discovery unless it fails in some important particular to conform with the previously recognized type, or until some other way is known of identifying the spirochete than by its morphological characteristics.

HISTORICAL AND GEOGRAPHICAL.

That relapsing fever existed was made plain by the British observers of the 18th and the early part of the 19th centuries, especially by Henderson (1843), Wm. Jenner (1849), and their contemporaries, and was so well differentiated from typhus and typhoid fevers that Clymer was able to recognize it in this country in 1844, and Griesinger was able to report that most of the cases of bilious typhoid in Egypt in 1851 were in reality relapsing fever. Later, when in Europe it was seen in epidemic form, it was supposed to be a disease indigenous to the British Isles.

Obermeier's demonstration, which gave the requisite criterion whereby the disease might be known in sporadic cases and variant types, was soon confirmed by Carter in India, who was the first Englishman to find the organism and to produce the disease in animals (monkeys).

So that there are two important periods in the natural history of relapsing fever. One may be said to begin with Henderson of Edinburgh, in 1843, and the other period may be said to begin when Obermeier in 1873 announced the discovery, which he made in 1868, of the organism which bears his name.

British Isles.—The years 1868–71 mark the last appearance of this disease in the United Kingdom, at least in epidemic dissemination. For 15 years before that it had not appeared. Moore, writing in 1891, says he had never seen a case, though he had seen service in two hospitals in Dublin for nearly twenty years, one of them being a large epidemic-fever hospital.

Whereas before Great Britain and Ireland had been an epidemic center, the disease has ceased to prevail there now for nearly forty years.

Russia.—Eastern Europe has had many visitations, especially Russia. It is said to have occurred first in 1833 at Odessa and in 1840 at Moscow (Hirsch, Dehio). A great epidemic raged in St. Petersburg in 1863–65; in Livonia, one of the Baltic provinces, in 1865 and again in 1883–84; Finland, 1865, Siberia, 1866, and Poland, 1868 (Hirsch), and smaller outbreaks over Russia in 1863–68 (Dehio). Clemow, quoting Reitlinger, of St. Petersburg, says it has reached as far north as Archangel, that in 1874 St. Petersburg was the great center, and that yearly reports came from Vologda, a northeastern province which had been formerly free. It existed in St. Petersburg in 1879–82, 1885–86, and 1890–91; Novgorod, 1898; it was rife in Warsaw in 1879 and 1883, in Moscow in 1882, and again in 1894–95 and 1898.

The observations of Gabritschewsky, of Moscow, on the development of immunizing substances (1896) may be mentioned here, Löwenthal's serum-diagnosis (Moscow 1898), and Sawtschenko and Melkich, also on immunity, in the province of Kasan on the upper Volga in 1900.

In 1893–95 the largest number of cases were reported from near the Volga in the southeast, and the smallest from the Baltic (Clemow, Dehio). Odessa has, from first to last, often been an epidemic center. Moschutkovsky of Odessa, 1876–79, produced the disease in healthy man by the inoculation of infective blood. Tictin, writing of the cases occurring in Odessa about 1889 or 1890, says relapsing fever occurs about once in every 15 or 20 years there.

The United States consul-general's office reports the total number of deaths in Russia from relapsing fever in the year 1901, as given by the Medical Department, as 2,466, out of a total of over

700,000 deaths from infectious diseases (including therein 250,000 from diarrhea and dysentery).

In 1905 the disease continued to figure in the mortality records of St. Petersburg, Moscow, and Odessa. The *United States Consular Reports* state for the week ending May 6, 4 deaths and 19 cases in St. Petersburg; for the three weeks ending May 13, 7 deaths and 126 cases in Moscow, and for the fortnight ending May 13, 1 death and 81 cases in Odessa, and yet, as the *U. S. Public Health and Marine Hospital Service Reports* point out, the malady has not been introduced into this country notwithstanding the large immigration.

Germany has been infected principally in the eastern part and probably from Russia (Hirsch). Upper Silesia, according to Murchison, showed cases in 1848, concurrently with typhus. The most important visitations, however, were those that took place from 1868–73 through northeast and south Germany and which gave rise to the brilliant work of Otto Obermeier in Berlin. Appearing first in Silesia in 1867, the disease spread the next year to Berlin, Stettin, Posen, Königsberg, and other cities. Again in 1878–79 other small epidemics occurred in Berlin, Dantzig, Magdeburg, Halle, Dresden, Breslau, and Würzburg, Heidelberg, and Giessen —1879–80. Since then Germany has been free from epidemics.

In 1895, however, there appeared an imported sporadic case, which is especially noteworthy because of the possible bearing it has on the mode of infection in view of the recent work on human tick-fever in Africa. This case was discovered in Hamburg among the emigrants by Dr. de la Camp, who says it is the first case that had occurred in the Emigrant Bureau Hospital.

De la Camp's Case.—The patient, a Persian on his way to the United States, entered hospital July 15, 1895, on the fourth day of his illness. Seventeen days previously he had started from Ooroomeyeh [a city of about 25,000 people, in the northwestern part of Persia, 64 miles southwest of Tabreez. The interest of this lies in the fact that the tick *Argas persicus*, which is said to cause disease in man, is principally found in Persia, in the neighborhood of Miana, a place 80 miles southeast of Tabreez]. The journey was made by horses to Tiflis; rail to Batoom, and thence to Hamburg by rail (steamer Batoom to Odessa).

On admission the temperature was 40.4° C., pulse 100; three paroxysms occurred of eight, six, and three days' length, respectively, intermissions of five, and thirteen days' length. Fever in first relapse developed gradually during three days, and during the relapses was quite remittent. Pulse during first and second fever periods was below 100, except twice 120 was noted; during third pyrexial period 120 to 160. Crises

were acute with marked sweats—greatest fall in second, 41° to 34° C., pulse 125 to 64. Each paroxysm was accompanied by headache, and muscle and joint pains, constipation, slight jaundice, enlarged and tender spleen, and a trace of albumin in the urine near the time of crisis, these symptoms disappearing during the intervals when the temperature became subnormal for a day or two. Spirochetes were discovered one hour after the rise of temperature in third paroxysm (12 to cover glass)—not found after crisis. Leucocytosis marked, but less during relapse and absent during intervals. Hb. 55 per cent. The case is reported in full.

Austria.—Relapsing fever, recognized first as bilious typhoid at Cracow in 1847, occurs nowadays especially in Bosnia and Herzegovinia. In 1889–90, in the latter province, an epidemic is reported upon by Karlinski at Stolac. This observer made some investigations on the transmission of this disease by bugs. The disease was epidemic in these parts in 1903. Hödlmoser, of Sarajevo, Bosnia, has done some work on serum diagnosis.

In the *Balkans* the disease is not frequently referred to, and according to Clemow in recent years has been absent from Constantinople. The well-known description by Hippocrates, in which he seems to allude to relapsing fever in the island of Thasos, off the coast of Salonica, may be referred to here, and it may be noted that he gives four symptoms occurring at the crisis, namely, epistaxis, eneuresis, dysentery, and sweating, that are of good omen.

The disease is said by many to be found occasionally in Greece, the Ionian Islands, and the Ægean Archipelago—Crete also, and Cyprus—in which places it goes principally under the name of bilious typhoid, which Griesinger pointed out and later Engel proved to be often really relapsing fever.

Asia Minor, Palestine, and Syria.—Cases are sometimes discovered in this part of the world. Tictin, at Odessa, in 1889 or 1890, saw a case in a sailor who had just arrived from a voyage which included a stay at Jaffa, where he seems to think the infection originated. Jaffa lies 31 miles northwest of Jerusalem. As the voyage was of some length, however, the infection might have originated on board ship. The latest record is by J. C. Cropper, at Jerusalem, July 15, 1905, who found a spirochete in the blood of a boy 15 years old who had never been out of the country. The boy was reported sick with a "fever of a quartan type." His temperature was 101° F. and he was not very ill. The organisms were not very

numerous ($\frac{1}{12}$ immers.). G. C. Low, of London School of Tropical Medicine, who examined the slides, reported that the spirochete was probably *Sp. Obermeieri.* Cropper says it is the second case he has seen.

Clemow says relapsing fever was never prevalent in *Transcaucasia.* Isolated cases occurred in 1889–90 in the city of Tiflis. Quoting Pantiukhoff, who wrote in Tiflis in 1898 on the "Statistics of Caucasian Pathology," he says that sporadic cases occurred along the railroad during the years 1894–98.

Of Mesopotamia and Persia, little or nothing is recorded. The disease is not uncommon in Western Siberia in the Government of Tobolsk.

India.—The disease has long been recognized in all parts of India and constantly recurs. The most important epidemic, from a historic point of view, was that which occurred in Bombay and its neighborhood in 1876–78. It was then that Carter made his important announcement confirming the presence of *Sp. Obermeieri,* and stating that "spirillum fever" is identical with relapsing or famine fever or typhus recurrens; that it can be readily communicated to the monkey (*Macacus radiatus*) and is "unequivocally manifested by both marked general symptoms and abundance of the attendant blood parasite," and that "postmortem appearances are equally concordant."

In a letter to the *Lancet* in March 1880, he made the important statement that he had induced spirillum fever in monkeys by inoculating with infected blood taken during the prefebrile incubation period. Seven years after the close of this epidemic Carter saw five undoubted cases of relapsing fever with Obermeier organisms in the blood, that is from September, 1885 to April, 1886, and by January, 1888 was able to report six cases more, proving, as he says, the continued presence in Bombay of this typical disease, and that notwithstanding the general prosperous condition of the local population.

In 1899 relapsing fever was discovered by Waters in the Common Prison at Bombay, the cases having been first reported as plague. The diagnosis was made by blood examination. Cuthbert Christy reports finding in September, 1900, while "at Ahmednegar, perhaps

the healthiest and nicest station in the Bombay Presidency," in cases of an epidemic disease in a near-by village, *Sp. Obermeieri.* In this same village plague had occurred the previous year. The disease, he adds, is endemic in certain quarters of Bombay. He made some observations at this time on infection by bedbugs, of which there are plenty there.

There can be no doubt that spirillum fever, to use Carter's term, is continually present in Bombay. Clemow (1903) says the disease, relapsing fever, was "severely epidemic during the last three years." A. Powell, of the British army, expresses the opinion that there must have been "tens of thousands" of cases in Bombay in 1901–2, and adds that it is strange it is not imported into Europe, the journey to London requiring but 14 days.

Amongst the hill tribes of the Himalayas it has long been known. According to Murchison it is said to have been recognized in 1852 in the valley of Peshawur, and again Hirsch shows that it was probably introduced into the Punjaub by the muleteers returned from service in Abyssinia in 1866. In the Kumaon Hill in 1898, Leon Rogers reports relapsing fever, and finds the *Sp. Obermeieri* in one of a group of cases all having similar symptoms and reported as mild plague by the natives. Also G. Browse reports finding this organism in a case simulating malaria occurring in March, 1904, at Nowshera in the Punjaub, in a well-situated and healthy regimental station. The patient was a cavalryman who had not been away from his station for a year. The case, he says, was exactly like cases diagnosticated as malaria in the Punjaub.

L. A. Walker found the parasite in some cases of a recurrent fever epidemic in the eastern portion of the Mardan district on the right bank of the Indus in 1904.

As to *Farther India and Malay Peninsula* the only mention I have found is that of Dr. James Kirk of Singapore, in a paper read before the Malay Branch of the British Medical Association, Session 1903–4, analyzing 150 cases of local fever. He says: "I have not yet diagnosed relapsing fever here."

East Indies.—A case of relapsing fever is reported in May, 1901, from Sumatra, by J. C. Graham at Deli, in which the spirochete was found. The patient was an immigrant Chinese coolie from

Swatau. He says it is the first case ever reported in Sumatra. The symptoms were high fever, severe headache, pains in the legs and joints, tenderness in both hypochondriac regions, slight icterus. No malaria parasites were present, specimen teemed with "*Sp. Obermeieri.*" Crisis on sixth day with fall to 35.6°.

China.—In the north, according to report, various foci existed in times past. In 1864–65 this disease was epidemic concurrently with typhus fever, and Tien Tsin suffered greatly in 1877. It was reported in the province of Shan Tung as very prevalent in 1889 (Neal—no blood verification).

In the south, the most common form of fever at Ting-Chau-fu is relapsing fever, to quote Clemow again.

From the province of Kwang Tung on the southeast coast spirochetic infection in sporadic instances is recently recorded from two places. One of these cases, namely Swatau, was mentioned above. The others are by L. G. Hill, of the Church Missionary Society Hospital, at Pak-hoi on the gulf of Tong King. In July, 1903, in making routine blood examinations of all cases, he discovered in a woman 65 years old "*Sp. Obermeieri.*" She had had a fever period of 11 days, an intermission of three days, and fever again for four days according to report, when admitted. A crisis occurred the same night. An interval of nine days followed, then a period of three days' fever, a one-day interval, and a one-day relapse. The organisms were many and active when first found. During the first two days of the first interval that was observed, the organisms continued in diminishing numbers and then disappeared till next paroxysm, when they were again plentiful, but not so active. Though the case was treated in the general ward, no other case developed.

On March 7, 1904, a Chinese coolie, aged 33 years who had been in the employ of a European for several years and who lived in the coolie quarters, was admitted with a temperature of 101°, having been ill several days. Myriads of parasites were found. No malaria organisms—excess of leucocytes. Crisis occurred two days later, the temperature registered 99.8° and no spirochetes were found. March 10, subnormal temperature and no organisms.

Hill says the disease had not been previously met with at Pak-hoi nor at Hong Kong.

In January, 1905, Koch of Hong Kong met with three cases among Chinese emigrants en route from Chin-wan-tao to South Africa. The periods of these cases were as follows:

Case I, two paroxysms of seven and four days with five days' intermission. Case II, fever 12 days, no "remission;" Case III, fever 12 days—sudden death after three days of apyrexia. Numerous "spirilla were found and no malaria." He states he is unable accurately to determine the duration of the first paroxysms. In the fatal case "no spirilla were found in any of the organs or in the blood of the heart. The spleen was enlarged and firm."

As a sequel of these cases the office boy of the hospital fell sick of the disease and went through a typical and severe attack with fever periods of six and ten days, separated by an interval of five days.

This observer says the disease is very rare in Hong Kong.

The Public Health Reports of the United States Marine Hospital Service for June 2, 1905, say that cases occurred in various places in China in the first part of last year.

Japan.—Baelz suggested in 1898 (as quoted by Clemow) that an epidemic disease which he observed in the island of Skikoku in 1881, may have been relapsing fever. Scheube (1903), who was formerly connected with the University of Kioto, does not speak of this disease as occurring in that country.*

Philippines.—I have been unable to obtain verification of the statement of Osler (1905) that these islands have suffered from several severe outbreaks—neither in Hirsch, Davidson, Clemow, Scheube, nor elsewhere.†

Egypt.—After the clinical demonstration by Griesinger, and the microscopical proof given by Engel at Cairo in 1884, we hear little of relapsing fever in Egypt till 1904. Clemow, writing in 1903, says the disease was formerly endemic in Egypt, but that it is absent from reports throughout Africa in general.

A good clinical account of this affection as it is seen in the Delta is given by F. M. Sandwith, of Cairo, in May, 1904. Sandwith found the spirochete in cases reported as typhoid in 1884 in and about Cairo and at Zagazig, an important cotton center 75 miles northwest of Suez. It appears that the disease has been more or

* One or two papers were published at Tokio in 1895 on recurrent fever, printed in Japanese.

† The report of Assistant Surgeon B. L. Wright, U. S. N., in the *Phila. Med. Jour.*, 1901, 7, p. 301, of cases of "Cavite Fever" puts one strongly in mind of relapsing fever. Although no reference is made to this disease in the article it is possible the cases were of that nature.

less continually present ever since. Eight deaths occurred in the provincial prisons in 1902. During the previous 11 years he had altogether 35 cases in his wards in Cairo. Suez, on the highway to the East, is another important center of distribution. Sandwith says the only case he ever saw in a European was at this place in 1892. In July 1905 there is a report of five cases at Cairo imported from Suez, in which the spirochetes were found by L. Phillips. The temperature charts are given. The shortest relapse was three days; the longest interval 10 days.

Abyssinia is noted above in connection with the Punjaub.

Tripoli, Tunis, Algeria.—Along the north coast of the African continent the disease has been reported recently by several of the French army surgeons.

A. Billet reported a case in an Algerian soldier, aged 30 years, in September, 1901.

> The man came to the hospital at Constantine with the diagnosis of malaria. He had two paroxysms: the first lasted four days; the second one and one-half days; the interval lasted 13 days. A few "Obermeieri" were seen the first day (four or five to field) and they increased to 10 or 15 to field before crisis. There was a prompt disappearance at the crisis. In the relapse a few were found (1 in 10 or 15 fields). A leucocytosis of 26,600 when the temperature was 39° C. on third day: polymorphonuclears, 77 per cent, large mononuclears, 1 per cent, small mononuclears, 22 per cent, eosinophiles, 0.1 per cent, 100 cells counted. The reds numbered 2,511,000. Hb., 85 per cent (Henaque). After the crisis the white cells fell to 19,000 (65 per cent polymorphonuclears) and in second paroxym, the count was 17,000 (74 per cent polymorphonuclears). Other features were absence of prodromes, slightly remittent temperature, rapid pulse (120 to 125) and respirations, enlarged spleen, some icterus, dry tongue, delirium, epistaxis preceding crisis, abundant urine and profuse sweat at crisis, followed by subnormal temperature.

Billet says the disease is excessively rare in Algeria. A small epidemic occurred near Constantine in 1866. Friant and Cornet record some cases occurring in Algeria near the frontier of Tunis in December, 1903 and January, 1904.

Lafforgue reports in 1903, from the vicinity of Tunis, 20 cases in which a spirochete was found identical in form and mobility and other characteristics with *Sp. Obermeieri.* He thinks cases probably occur in Tripoli.

Morocco.—It is said by both Davidson and Clemow to have appeared in this country during the periods of famine following plagues of locusts.

Grand Canary.—In the *U. S. Consular Sanitary Report* for the week ending May 6, 1905, 13 cases of relapsing fever were reported in the Grand Canary (Spanish).

Gibraltar.—Here may be noted the case of infection by spirochetes that was reported by Manson in 1904. An English lady, aged 30 years, who, having resided at Gibraltar for three years, returned there after a short visit to England on September 29, 1903. On October 20 she had a rigor followed by a fever (104° F.) which after profuse sweat terminated in four days. A ten-day intermission and a second paroxysm occurred, lasting three days, after five days a third, lasting also three days. Another interval of 10 days, during which time the patient returned to England, and on November 24 a fourth period of fever began. After this paroxysms occurred on December 4, 15, 29, and January 11. Each paroxysm except the last, which was very mild, was ushered in by rigor and terminated with profuse sweating. Spirochetes were found in the paroxysm of December 29, but were not looked for before, except during the subsiding fever of the previous period. They were few in number, and Manson is noncommittal as to whether it was the *Sp. Obermeieri* or not. He thinks there were too many relapses and that the spirochete (of which an illustration is shown) differs in shape from this well-known organism.

The number of paroxysms is greater than in any other case I have found record of.

Tropical Africa.—In the course of the years 1904 and 1905 several clinical and three most important experimental reports were made under the various titles: relapsing fever, spirillum fever, spirillosis, and "tick-fever" from British East Africa, the Congo Free State, German East Africa, and Portuguese West Africa.

Uganda.—A. R. Cook, from the missionary hospital at Mengo, at the head of Lake Victoria Nyanza, published, early in 1904, some notes of cases of relapsing fever.

Case I.—March 21, 1899. A little girl, whose blood teemed with spirilla, was admitted in extremis and died 16 hours later; no postmortem examination. During the next four years isolated instances were seen, and in November, 1903, a somewhat widespread epidemic was noted.

Case II.—November 7, 1903; a man ill three days with vomiting and pains in the chest; no rigor; no diarrhea; collapsed condition; temperature 99.4°, pulse 140, respiration 80; conjunctivæ jaundiced; delirious. Spleen and liver not felt; very

scanty urine, trace of albumin. Fresh specimen of blood showed over 30 spirilla to one field ($\frac{1}{12}$ immers., oc. III); death in four hours. Autopsy three-fourths hours later. Smears from the enlarged spleen showed few spirilla.

Case III.—November 8, 1903. A man three days sick in his second paroxysm. Rigor at the onset. No vomiting nor pains. Temperature 101.6°, rising in one hour to 104.2°; slight delirium; no jaundice. Blood showed one organism to each field. Crisis on fourth day when spirilla disappeared.

Case IV.—November 15, 1903. Small girl whose blood swarmed with spirilla Temperature 105°; no accurate history.

Nabarro, while studying sleeping sickness in Uganda, met with a case of spirillar infection on August 1, 1903.

Hodges and Ross, also in Uganda, discovered the organism in the blood of an Indian who had been absent from India for over a year and who had never had a similar attack before. These organisms were few, sharply pointed at both ends, and were about 40 μ long and 4 μ broad. The symptoms were vomiting, back-pain, temperature 103.4°, with spleen slightly enlarged and tender. Crisis on the next day; relapse 18 days later when spirilla were again found. A monkey was inoculated. After three and one-half days fever began in the monkey, lasting three days; numerous spirilla. Every case of fever was examined (60) coming from various parts of the Protectorate, and 12 showed spirilla, comparatively few organisms in all. In the one fatal case the spirilla disappeared from the blood 24 hours before death, while the temperature was still high.

Angola.—Portuguese West Africa is a province bounded on the north by the Congo State. From Benguela, a district on the coast of this province, in the early part of last year A. Y. Massey reported a case of spirillosis in a man which he met with in December, 1903. The patient, a Portuguese trader, had a temperature of 103.4° and involuntary passages, and died three days after admission. Fresh specimens of blood showed spirilla innumerable. Next, Wellman, in April, 1905, reported from Angola a case of relapsing fever with spirilla. The patient, a native 32 years old, was admitted March 3, 1904, one day sick; temperature 104.4°, pulse 92, respirations 29. No rigor or vomiting; organisms about a dozen in each field ($\frac{1}{12}$ immers., oc. II). Crisis occurred on the third day. A few spirilla persisted for two days more. After an intermission of seven days a relapse of three and one-half days occurred during which the organisms were fewer in number.

Ile de Réunion and Mauritius.—These islands off Madagascar were infected with relapsing fever in the year 1865, the disease having been introduced direct from India by coolies. It lasted for several years (Hirsch and Stephen Smith).

Uganda (*Tick Fever*).—Continuing the investigations of Hodges and Ross at Entebbe, Uganda, Philip H. Ross and A. D. Milne, knowing of Marchoux and Salimbeni's demonstration that spirillosis of fowls is conveyed by a tick, thought that perhaps the so-called tick-fever might be due to a spirillum. They were unable to get any reliable description of symptoms, nor good temperature records. Some cases had vomiting and some had relapses; some had splenic tenderness. The most common symptoms were severe headache, pains in back and limbs, and fever ending critically. All had spirilla sparsely scattered and difficult to find. They concluded that certain cases diagnosticated as tick-fever are due to a spirillum. Their observations were published on November 26, 1904. On the same date J. E. Dutton and J. L. Todd cabled from the Congo: "Spirilla cause human tick-fever. Naturally infected ornithodoros infect monkey."

Congo Free State.—Dutton and Todd were working independently of Ross and Milne and were on the Upper Congo at Kasongo some distance west of Lake Tanganyika in November, 1904, when they both contracted this disease which later led to the death of Dutton, February 27, 1905. A report of their work was issued last November (1905) and their conclusion is that the tick-fever in the oriental province of the Congo Free State is a relapsing fever, produced by a spirochete, probably identical with *Sp. Obermeieri*, and that this organism can be transmitted by the tick *Ornithodorus moubata.* The report contains records of 14 cases of which two cases were Europeans, Dutton and Todd themselves being the victims. There are also notes of animal experiments, and remarks on the distribution of the human tick in the Congo region, together with notes on the external anatomy of *O. moubata* by Newstead.

Dutton and Todd remark what Livingston said, that the ticks followed the Arabs, and point out that the ticks seem to have come into the Free State by two routes; from the East Coast with the Arabs, and with traders from the Portuguese territory to the south.

A closely allied, if not identical, ornithodorus is found in Angola (Newstead).

Cuthbert Christy found these ticks in Uganda in 1902, and says that they are distributed in German East Africa, Angola, and in the Zambesi, and are closely allied to the *O. Savignyi* found in Egypt, Nubia, Abyssinia, and Somaliland.

Milne says that one of the chief caravan roads in German East Africa, south of Victoria Nyanza, had been closed, owing to constant sickness caused by tick-fever. Koch found the ticks widely distributed over the district (1905).

The tick *Argas persicus* is found in Persia and Beluchistan, being especially common, according to Manson, in the northern part of Persia in the neighborhood of Miana. The natives attribute a disease to its bite, which is known as the "disease of Miana." (Compare de la Camp's case noted above.) Manson quotes Schlimmer as saying that on one occasion (1858) he treated 400 soldiers who declared they had been bitten by these ticks at Miana. The symptoms resemble those of "remittent fever—extreme lassitude, disinclination to work, yawning, fever, perspiration, not accompanied by much thirst, increasing and decreasing at stated hours of the day."

United States.—The first introduction of relapsing fever into this country was by Irish immigrants in 1844 at Philadelphia, when Clymer was the first to recognize it, he having 15 cases under his his care. Pepper, Parry, and others also saw cases. The disease did not spread far. In 1850–51 Flint differentiated cases from among some Irish immigrants affected with typhus, who arrived at Buffalo where he was then in practice, and he also reported 15 cases, and adds that doubtless others occurred which were mistaken for typhus. It is mentioned by Clymer that, in a report from the Russian government to the English ambassador at St. Petersburg, the disease was said to exist at New Archangel (now Sitka, Alaska) in 1858. The next and last time it appeared here was in epidemic form in New York. The first case appeared in October, 1869. The epidemic lasted through the winter and spring and in rapidly waning numbers into 1871. In Philadelphia numerous cases appeared. The disease did not spread far from these centers, however. A few

cases occurred in Washington, in Maryland, New Jersey, and Connecticut, and one case was imported into Boston, as noted by Shattuck in the American translation of Strümpell's work on "Practice of Medicine."*

In September, 1874, a severe outbreak of epidemic disease was observed at Oroville, California among Chinese laborers. Many of these cases were similar to typhus, judging from the description, but many had the characteristics of relapsing fever; crisis on the sixth day and relapse on the 14th day after a period of complete apyrexia, and are so reported by the observer, Miller.

At Worcester, Mass., in August, 1899 a sporadic case appeared in an Armenian immigrant and is recorded as relapsing fever by Ward. This diagnosis was not confirmed by blood examination, though the spirochete was looked for during the relapse. The case presents all the other characteristic features, however. Malarial organisms were absent.

South America.—Baldou says it appeared in Peru in 1854 and, known as "peste des Cordillères," spread thence slowly to Chili and Bolivia (1856). Hirsch (1881) says nothing is known of its existence in Central and South America.

Cuba.—Relapsing fever is next heard of in this part of the world in 1902, when Biada, of Havana, reports, under the title "recurrent fever," an important case that simulated yellow fever and by a careful examination of the blood was proved to be due to spirochetic infection.

Biada's case.—Spaniard (Espanol) aged 19 years, a sailor for one and one-half months on steamer from Liverpool, to the coast of "Cantabricas"(Spain?), to Cienfuegos, to Havana, and then on a coasting schooner. The onset was abrupt on January 19, 1902, with chill. Temperature 40° C., pulse 116, respirations 30, pains all over, especially in legs; constipation; spleen very large. No malaria parasite was seen in the blood, but perfectly characteristic *Sp. Obermeieri* (three in one field) were discovered by Dr. Kirby Smith. The urine (550 c.c.) contained some albumin on third day. The fourth day much pain in splenic region, diarrhea; fifth day, epistaxis; sixth day black vomit, slight jaundice, crisis—fall from 103° F. to 95.4° from 6 A. M.

* A case of relapsing fever entered the Presbyterian Hospital, New York, last July (1905) in the service of Dr. George A. Tuttle who kindly permits me to mention the fact. The patient, an Armenian woman who had been in this country for a number of years, had entertained, a few days before her sickness began, some relatives newly-arrived from Armenia; they themselves were not sick, however. The patient had two paroxysms of fever, each of five days' duration separated by an interval of like length. The diagnosis was confirmed by the finding of the organism in the blood during the relapse. The case is to be reported in the *Medical and Surgical Reports of the Presbyterian Hospital for 1905–6.*

to 6 P. M.; disappearance of spirilla. On the seventh day felt better; stools formed, urine 325 c.c., less albumin. Eighth and tenth days, urine between 1,400 and 2,200 c.c.; felt fine on 11th day; no spirilla. February 7, 13th day after the crisis, epistaxis returned urine contained much albumin, and again, six days later, no fever appearing and no organisms, abundant albumin was noted. February 20, slight iritis. He was subsequently discharged without having any relapse of fever, but with some edema of extremities, a little albumin in the urine, and bad color to skin.

The day before the crisis (January 24) a monkey was subcutaneously inoculated with 3 c.c. of patient's blood. Spirochetes were found in the monkey's peripheral circulation 61 hours after inoculation. The animal's temperature ranged between 40° and 39° C. for two days and fell on third day coincidently with the disappearance of spirochetes.

Mexico.—Some cases were noted here in April, 1905. The Surgeon-General's office of the U. S. Public Health and Marine Hospital Service writes in answer to an inquiry, that they were reported from Tuxpam, that deaths occurred from this disease during every month throughout the year, but that the weekly sanitary reports from United States Consul Lesspinasse at Tuxpam do not state the number of cases occurring, nor give other information relative to the deaths that have occurred. Tuxpam is a seaport on the gulf coast between Vera Cruz and Tampico—145 miles northwest of the former place. It has a large trade in cedar, vanilla, fustic, and coffee.

Panama.—The Surgeon-General has very kindly furnished the following statement, dated February 14, 1906: "It is understood that two cases of the disease (relapsing fever) were under treatment in the Colon Hospital, Colon, Panama, during the summer (1905), the diagnosis having been verified by blood examination."

The foregoing account shows that there are cases of periodic fever occurring in many widely separated parts of the world which, in their clinical manifestations and infective character, are strikingly similar to relapsing fever, and, as regards the associated microorganism parallel, if not identical, therewith. In respect of so-called tick-fever, Dutton and Todd, in their report, are as explicit as Carter was. They say (p. 4): "The case reports, charts, and postmortem finding certainly demonstrate the clinical identity of the tick-fever observed by us, with the relapsing fever of the textbooks." Koch says that the relapsing fever seen by him in 1905 in German East Africa is closely similar to the European relapsing fever, the unlikeness being

that the relapses are shorter and the spirochete is, as a rule, longer in the African disease.

MODE OF INFECTION.

Most, but not all, of the older writers believe that relapsing fever is a contagious disease.

Many authorities might be cited and many instances given to show that the disease is not a highly contagious one; that, on the introduction of a case into an uninfected area, the disease does not spread rapidly nor far; that from the existence of a single case in cleanly surroundings, and without special attempts at isolation, the disease is not newly developed.

None of the reasons given, tending to prove that the disease is a contagious one in the sense that typhus fever is said to be, is inconsistent with the view that it is conveyed by blood-sucking insects, such as have been proved to be the means of infection of spirochetic disease, as in so-called tick-fever in man, *Sp. Theileri* in cattle, spirillosis of fowls, etc.

Nuttall says that Flügge, in 1891, was the first to suggest the possibility of vermin serving to spread relapsing fever. Tictin, in 1897, at Odessa experimented with the ordinary bedbugs, and succeeded in causing the disease in monkeys by inoculating them with blood taken from bugs immediately after they had fed, but failed to succeed when an interval of 48 hours was allowed to elapse.

Karlinski found abundant spirochetes in bugs caught in houses in Bosnia, where relapsing-fever cases existed, and none in bugs caught in other houses free of this disease. He says that spirochetes persisted for 20 days in bugs. Dutton and Todd found living spirochetes in the stomach and malpighian tubules of ticks up to five weeks after their feed on a known infected animal, and were able to infect a monkey after one and three-fourths months. One of of their conclusions is that the transmission (of the spirochete) is not mechanical, but some developmental process is carried on in the tick.

Schaudinn suggests that certain stages in the life-history of the *Sp. Obermeieri* may be formed in the blood of the patient which in relapsing fever are not recognized as parasites.

Cuthbert Christy at Ahmednegar, India, did not succeed in acquiring the disease by allowing himself to be bitten by bed-bugs caught in the bed of a patient sick with relapsing fever.

In Case I of this report, the disease undoubtedly originated outside of New York. That spirochetic disease existed in the part of the world where this patient has spent much of his time recently has been shown—Havana, 1902, Colon, 1905, and perhaps cases at Tuxpam. No case developed in Bellevue Hospital subsequent to his stay of 89 days in hospital, and absolutely no special means were taken to prevent infection. There are several varieties of ticks to be found in the West Indies, South and Central America (*O. talaje*) and Mexico (*O. turicata*). Sambon has suggested the *O. turicata* in Mexico as a posssible factor.

DIAGNOSIS.

There is great approach to unanimity shown by the various observers in the terms employed, namely, relapsing fever, recurrent fever, recurrent typhus or spirillum fever. Furthermore, a study of all these reports and our cases leads one to the conclusion that these cases cannot be differentiated one from another either clinically, or by the morphological features of the spirochete, as far as these are known, or by animal inoculation.

A thorough search of the blood during the pyrexial period, and according to Carter, even during the pseudo-crisis, will reveal the spirochete. These are not very numerous in many of the cases. They increase in number with the progress of the febrile period, and in each succeeding relapse, because of decrease in numbers, more care is required in the search. They begin to disappear just before the crisis, but may be found (in Case I in four large smears and in one of Hill's cases at Pak-hoi and the first two cases of Dutton and Todd) a day or two after the fall. The statement of Sandwith, that the spirilla entirely disappear from 12 to 24 hours before the temperature falls at the crisis, is certainly not true for most of the cases, though Heidenreich, of St. Petersburg, quoted by Fagge, makes a somewhat similar statement. Heidenreich mentions what has been spoken of by many of the recent observers, that there are fluctuations in the number of the organisms during the same

pyrexial period, and differences in mobility, size, and shape may be noted in the same specimen of blood (Carter, Dutton and Todd, and others, and see accompanying microphotographs from the same smear of blood in Case I). Carter and Heidenreich have found the spirochetes during the interval, just before the relapse, and in Cases 8 and 9 of Dutton and Todd the day before the rise of temperature. They were seen in the interval also in one of the native "tick-fever" cases, by Ross and Milne—the patient complaining of severe headache, but without a coincident return of fever. Sometimes when a relapse is about due, the patient complains of some subjective symptom, headache, wakefulness, restlessness, and the temperature is slightly raised (subfebrile), but no spirochetes are found. (Fagge and Cases I and II of this report.) The organisms have been found after death, in the blood and in the spleen (Strümpell, Dutton and Todd's Case IV and Cook's Case II).

The blood, besides the positive proof presented during the febrile period, shows some changes in the leucocytes which apparently vary in greater or less degree, side by side with the progress of the disease.

Many of these cases, in which the number of white cells are recorded, show a moderate leucocytosis (de la Camp, Hill, Billet) particularly distinct in the first paroxysm. Case I of this report, and of the three instances of Dutton and Todd's report in which the note is made, Cases XIII and XIV, do not agree with this, in the other of these three instances, the fatal case, the count was 14,200 and during the crisis 11,800.

It may be said that there is very often recorded, in the first paroxysm, an increase in the number of white cells, particularly the polymorphonuclears, which diminishes early in the interval and is less marked with each recurring paroxysm. This is not invariable, as is shown in the table given above, in which the most marked change is in the relative number of large lymphocytes and mononuclears. No distinct changes in red cells other than those indicative of secondary anemia occur.

A study of these sporadic instances of spirochetic infection suggests that the following symptoms may indicate the probability that this condition is present.

A period of fever of four or five days' length, beginning abruptly and ending abruptly, chilliness or chill at onset, recurring just before the crisis. Sometimes epistaxsis at the same periods, very severe headache, backache, and leg pains. Moderate sweating and moist tongue during the fever. Moderate enlargement of spleen, with considerable tenderness on pressure. Pulse increased, perhaps rapid. Recurrence of similar paroxysms at any time from third to eighteenth day, usually between seventh and fourteenth. Relapses short, from one-half to three days as a rule, during which fever may be higher than in first paroxysm. The number of relapses may be from one to seven—perhaps more. Often a pseudo-crisis the day before the true crisis. Crisis accompanied by drenching sweats coincidently with a rapid and very great fall of temperature occurring during the night and extending over 12 or 16 hours.

The interval characterized by temperature and pulse-rate much below the normal standard; this often continuing until the day before the next paroxysm. Sweating persisting often for two days at the beginning of the interval; disappearance of splenic tenderness and quick return to feeling of well-being.

Löwenthal has proposed a serum reaction which (when the opportunity offers) he says will enable the diagnosis to be made during the interval. This reaction lies in the power of the blood serum of a patient who has passed through one paroxysm to agglutinate the spirochetes in the blood of a fever case, a power not possessed by normal blood serum.

Malaria is the disease for which these cases of spirochetic infection are most likely to be mistaken in this part of the world. In a patient presenting symptoms similar to malaria and who has recently arrived from foreign parts or who has been more or less intimately associated with a newly arrived person, a careful search of the blood should be made for this organism in question. It is perhaps allowable to point out that, compared with relapsing fever, malaria presents a more regular incidence of febrile periods; a shorter interval between the paroxysms, and generally a spleen more easily palpated.

The blood in malaria shows an early and a greater loss in the red cells and hemoglobin percentage. As regards the white cells, the importance of the presence of intracorpuscular pigment is to be

kept in mind. The leucocytosis observed by many in relapsing fever is unusual, if not wanting, in uncomplicated cases of malaria.

On the other hand, the normal or diminished white-cell count associated with a very high relative increase in the larger lymphocytes and mononuclear cells seen in our cases, has been seen by many in malaria (see Stephen's and Christopher's note quoted in Mannaberg's article in Nothnagel's *Enclyclopædia*).

Mannaberg quotes Mamourski as having seen a case which showed both a malaria parasite and the spirochete of Obermeier in the blood at the same time.

Influenza is suggested by the sweating and the marked subnormal range of temperature characteristic of the intermission. Rabagliati says he knows of no other diseases in which this feature is so apt to be emphasized as relapsing fever and influenza. In the latter disease a relapse, if it occurs, follows the first febrile period after an apyrexial stage of much shorter duration than that that rules in relapsing fever, and the symptoms in such a case do not promptly or entirely disappear on the fall of temperature.

Considering the fact that the vomiting of blood, so common in yellow fever, may occur in spirochetic infection (Biada's Case), and that epistaxsis and jaundice occur in the latter condition, one should always look for this organism in the blood of cases showing some of the characteristics of yellow fever. A statement similar in effect may be made of cases that suggest the presence of plague, typhus fever, or sepsis.

The duration of Case I was 30 days, of Case II, 25 days; Dutton's case lasted for 47 days, Todd's case for 42 days. Taking such an instance as Manson's Gibraltar case and adding an incubation stage of a week, we have a period of three months and in that length of time one of these cases might travel from the remotest parts. A sporadic case of spirochetic infection may, therefore, appear anywhere at any time, and undoubtedly it occasionally so happens.

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PLATE 6.

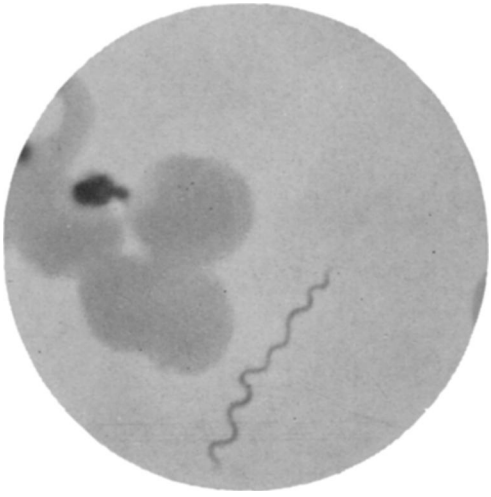

FIG. 1.

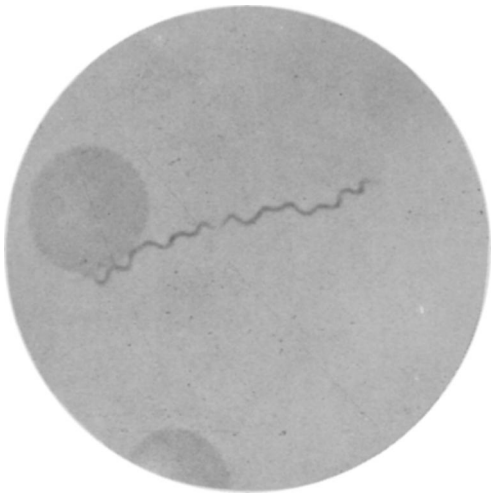

FIG. 2.

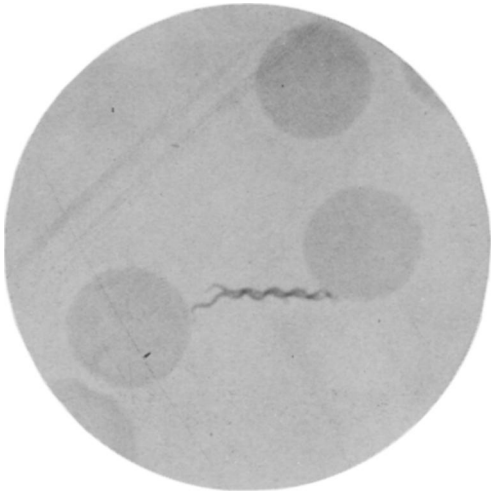

FIG. 3.

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EXPLANATION OF PLATE 6.

These photographs (1 500 diam.) were taken from one and the same specimen of blood from Case I.

FIG. 1.—A large spirochete.

FIG. 2.—Two spirochetes seemingly end to end.

FIG. 3.—Two small spirochetes intertwined.